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Right Ventricular Involvement in Obstructive Cardiomyopathies : Hæmodynamic Studies in 13 Cases

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Many terms have been used to describe the obstructive cardiomyopathies (McMichael, 1964). Most of them centre around the common, and apparently exclusive, theme of left intraventricular obstruction. The participation of the right ventricle is however described in numerous studies. Protrusion of the hypertrophied septum (Brent *et al.*, 1960; Soulié, Joly, and Carlotti, 1962), hypertrophy of the free wall (Livesay, Wagner, and Ambrust, 1960; Daoud, Gallaher, and Kaplan, 1961), and of the septal and parietal parts of the crista supraventricularis, are common anatomical findings (Braunwald *et al.*, 1964). The ejection systolic murmur at the pulmonary area (Wigle, 1964a), the radiological signs of right ventricular hypertrophy (Bevegård, Jonsson, and Karlöf, 1962; Braunwald *et al.*, 1964), the electrocardiographic evidence of right atrial hypertrophy, isolated or combined with that of the left atrium (Braunwald *et al.*, 1964), the large "a" wave in the jugular pulse tracing (Wigle, 1964a; Braunwald *et al.*, 1964) are all clinical clues suggestive of involvement of the right heart. Cardiac catheterization makes it possible to demonstrate a right intraventricular systolic pressure difference. Its true incidence, location, and degree have been variously interpreted. The obstruction is usually thought to be in the lower part of the outflow tract where it is found in 6 cases out of 8 (Soulié *et al.*, 1962), 6 out of 9 (Wigle, Heimbecker, and Gunton, 1962), and 10 out of 59 (Braunwald *et al.*, 1964). The infundibular site of the right intraventricular obstruction has recently been questioned, for the systolic pressure difference has been located between the apex and the outflow tract (Cohen *et al.*, 1964). This point has been emphasized by the systematic exploration of the apex of the right ventricle (Taylor, Bernstein, and Jose, 1964; Bourdarias *et al.*, 1964; Goodwin *et al.*, 1964).

It seemed interesting, therefore, to find out by catheterization and cine-angiocardiography of the right heart chambers, the location and frequency of right intraventricular obstruction in this condition, and to study the influence of pharmacodynamic agents and post-ectopic beats upon the obstruction.

Patients and Methods

Thirteen patients with obstructive cardiomyopathies have been studied. Cases 1, 3, 5, and 10 have been reported previously (Bourdarias *et al.*, 1964). The details are to be found in Table I. The usual mid-systolic murmur was heard in 11. The characteristic bulge was seen on the carotid pulse tracing in 11. Cine-angiocardiography displayed evidence of left ventricular obstruction (Ferrané *et al.*, 1964) in all 9 patients in whom it was performed. A left intraventricular systolic pressure difference was found either at rest in 6 patients or during isoprenaline infusion in 3 patients in whom the catheter could be manipulated into the apex of the left ventricle. Two sibs of Patient 10 were known to have the same condition. A group of 15 control patients has also been studied, and the diagnoses are listed in Table II.

The patients were catheterized in the morning, fasting, in the recumbent position after a light premedication by subcutaneous administration of 0·1 g. phenobarbital. Pressures were recorded by means of No. 6 or 7 F Cournand catheters connected to electromanometers*. The zero pressure reference plane was halfway between the catheterization table and the sternal angle. Attempts to manipulate the catheter into the apex of the right ventricle were successful in 12 instances. Pressures were recorded during withdrawal of the catheter from the pulmonary artery to the right atrium and from the apex of the right ventricle to the right atrium in 7 observations.

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*Chargé de recherche au C.N.R.S.

* Electromanometers TELCO, model M. 41. R.

TABLE I

OBSTRUCTIVE CARDIOMYOPATHY: PATIENTS STUDIED

Subject, sex, and age			Ejection murmur	Electrocardiogram	Bulge on carotid pulse	Cine-angiographic evidence of subaortic stenosis	Left ventricular-aortic gradient (mm. Hg)*
1	F	59	+	LVH		+	†
2	M	11	+	LVH–RVH	+	+	40
3	F	59	+	LVH	+		96
4	M	24	+	LVH–septal "necrosis"	+	+	7·5 (117)
5	M	44	+	LVH–ILBBB	+	+	27
6	F	32	+	Anterior "necrosis"	+		†
7	M	35	+	LVH	+	+	62 (146)
8	M	14	+	LVH–IRBBB	+	+	0 (122)
9	F	21	0	LVH	+	+	0 (75)
10	M	37	0	LVH–ILBBB Biatrial enlargement			34
11	M	35	+	LHV	+		63
12	F	19	+	LHV	+	+	†
13	M	12	+	LHV–ILBBB	+	+	†

LVH, RVH, left or right ventricular hypertrophy, ILBBB, IRBBB, left or right incomplete bundle-branch block.
* Figures in brackets refer to values of gradient during isoprenaline infusion.
† Failure to thread the catheter through the stenotic area into the substenotic chamber.

TABLE II

CONTROL GROUP: HÆMODYNAMIC DATA

Subject, sex, and age			Diagnosis	Systolic pressures (mm. Hg)				Systolic gradient apex-outflow
				Inflow tract	Apex	Outflow tract	PA	
			I: *Concentric hypertrophy of right ventricle*					
1	M	12	Pulmonary stenosis	64	66	64	16	2
2	M	11	Pulmonary stenosis	51	57	55	16	2
3	M	16	Pulmonary stenosis	61	61	61	25	0
4	F	20	Pulmonary stenosis	116	180	120	16	60
			II: *Diastolic overload of right ventricle*					
5	M	30	Patent ductus arteriosus; interventricular septal defect	22	22	21	21	1
6	F	23	Ostium primum	44	47	40	40	7
7	M	8	Patent ductus arteriosus	27	118	26	26	92
8	F	10	Atrial septal defect; mitral insufficiency	37	36	35	35	1
9	F	7	Atrial septal defect	28	29	28	28	1
10	F	32	Atrial septal defect; anomalous pulmonary drainage	29	28	28	28	0
			III: *Miscellaneous conditions*					
11	M	17	Aortic insufficiency	19	25	19	19	6
12	M	36	Polycythæmia	30	34	33	33	1
13	M	28	Non-obstructive cardiomyopathy	36	38	37	37	1
14	F	15	Right bundle-branch block	26	25	22	22	3
15	M	43	Aortic stenosis	26	30	28	28	2

The effects of isoprenaline infusion (4·0 to 4·5 μg./min.) were studied in Patients 6, 7, 8, and 9. Pressures were recorded between the tenth and the twentieth minute of infusion, once a steady state had been achieved. Intravenous propranolol (inderal)† (priming. 0.010 g., sustaining 150 μg./min.) was infused in 2 patients (Cases 11 and 12). Ectopic beats were deliberately elicited in observations 4, 10, and 12, in order to study the gradient during post-ectopic beats. In observation 10, pressures were studied during asynchronous pacing transmitted by a unipolar electrode catheter placed successively in the outflow tract of the left ventricle and body of the right ventricle. Cardiac output was measured by the direct Fick method. Oxygen uptake was measured by means of a ventilated spirometer* for three minutes. Blood samples were drawn from the pulmonary and brachial arteries during the second minute. Oxygen capacity was measured by the Van Slyke manometric method. Oxygen saturations were determined in a Brinkman Hæmoreflector†.

† ICI 45520 kindly supplied by Imperial Chemical Industries Ltd.

* Pulmotest Godard, The Netherlands.
† Kipp, Delft, the Netherlands.

TABLE III

OBSTRUCTIVE CARDIOMYOPATHIES—HÆMODYNAMIC DATA UNDER BASAL CONDITIONS

Subject	Pulmonary artery (mm. Hg)	Right ventricular pressures (mm. Hg)			Systolic gradient				Cardiac index (l./min.m.^{2})	Stroke index (ml./beat m.^{2})
		Apex	Outflow tract	Inflow tract	Apex-outflow	Inflow-outflow	Apex-inflow	Apex-pulmonary artery		
1	29/10	144 / 2 16	26 / 3 5	33 / 3 6	118	7	111	115		
2	32/15		31 / 1 5	36 / 2 5		5				
3	37/17	84 / 2 7	36 / 2 4	53 / 1 5	48	17	31	47		
4	16/7	32 / 5 9						16		
5	26/11	118 / 2 8	27 / 1 8	38 / 2 6	91	11	80	92	3·76	52
6	28/12	33 / 6 8						5	5·04	55
7	25/9	41 / 5 10	25 / −3 5	25 / −3 5	16	0	16	16		
8	27/9	35 / 1 10	28 / 0 7		7			8		
9	19/7	33 / 0 5	20 / 0 6	24 / 0 5	13	4	9	14	4·04	48
10	38/23	73 / 0 8	45 / 4 8		28			35	2·14	27
11	35/11	73 / 2 10						38	3·32	57
12	19/10	40 / −3 6	19 / −2·5 1	26 / −1·5 6	21	7	14	21	3·08	24
13	28/10	104 / 2 7	28 / 0 6	50 / 2 7	76	22	54	76		

Results

Right Ventricular Pressures in Basal State. Observations made during basal conditions are listed in Table III. A significant peak systolic gradient (greater than 7 mm. Hg) was found in 11 out of 12 patients between the apex of the right ventricle and the pulmonary artery. The pressure difference ranged from 8 to 115 mm. Hg, and was related to an obstruction between the apex and the outflow tract of the ventricle. This was demonstrated by the fact that in 9 of these 11 patients pressures were nearly identical in the outflow tract of the ventricle and in the pulmonary artery. A peak systolic pressure difference (range 9 and 111 mm. Hg) was found in the 7 in whom pressures were simultaneously recorded from the apex and inflow chamber of the right ventricle. On the other hand, a systolic pressure difference was only found in 3 out of 8 patients, between the inflow tracts of the right ventricle. In these 3 instances, this infundibular gradient was moderate as compared to the gradient found between the apex and the upper part of the right ventricle. These findings suggest: first, that an intraventricular obstruction exists during systole which separates a high pressure chamber at the apex and a low pressure chamber which comprises the inflow and outflow tracts of the right ventricle; second, that in some cases a less important systolic obstruction exists between the inflow and the outflow tracts of the right ventricle (Fig. 1, 2, and 3).

Three other anomalies were found in the pressure tracings recorded from the right side of the heart.

First, filling pressures were raised. End-diastolic pressure in the apex of the right ventricle was in all instances at or above 5 mm. Hg (Table III) and the "a" wave was always conspicuous (Fig. 1, 2, 4, and 5) and rose well above the end-diastolic pressure.

Secondly, the ascending limb of the systolic pressure recorded from the apex of the right ventricle was interrupted by a notch, after which the rate of rise in pressure slowed. The pressure at the time of the notch was close to (Fig. 2 and 4) or

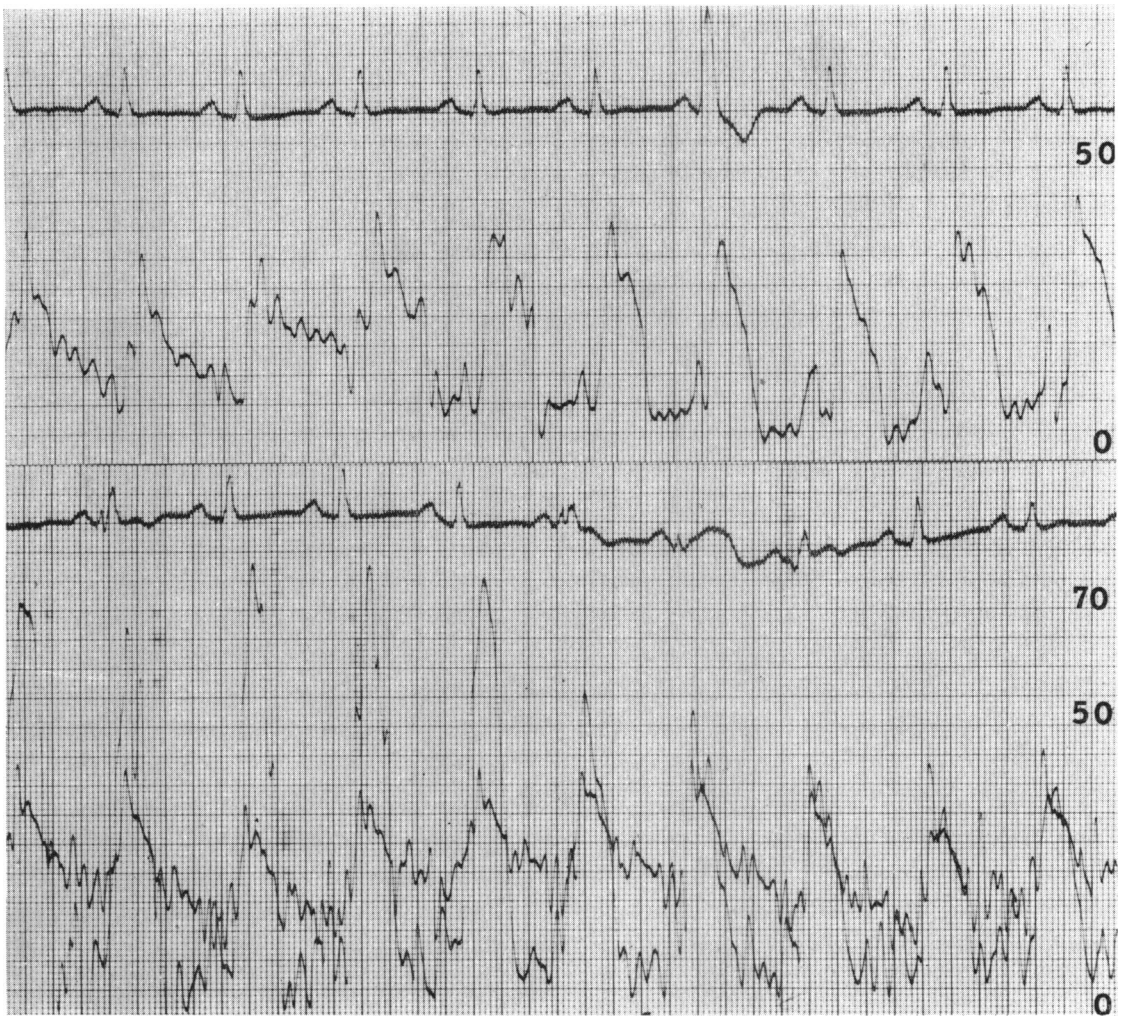

FIG. 1.—Case 7. Obstructive cardiomyopathy. *Upper half.* Pressure is recorded during withdrawal of the catheter from the pulmonary artery to the right ventricular inflow tract. There is no intraventricular pressure difference between the outflow and inflow tracts. *Lower half.* Pressure is continuously recorded in the pulmonary artery, while another catheter is withdrawn from the apex to the inflow tract of the right ventricle. There is a significant systolic pressure difference between these two parts of the ventricle. Paper speed = 25 mm./sec. Pressures in mm. Hg.

above (Fig. 1 and 5) the peak systolic pressure in the pulmonary artery.

Thirdly, the systolic wave in the pulmonary artery sometimes had an abnormal pattern: an early systolic peak was followed by a mid-systolic sharp trough which in turn was followed by a late systolic secondary pressure rise. These anomalies could be obvious (Fig. 4), doubtful (Fig. 5), or absent (Fig. 2).

Correlations Between Left and Right Intraventricular Pressure Gradients in Basal State. Fig. 6 shows the lack of correlation between the basal right and left intraventricular gradients measured simultaneously in 8 patients. The absence of a basal left intraventricular gradient when such a gradient exists in the right ventricle is not reliable evidence of isolated right-sided obstructive cardiomyopathy. This point is clearly demonstrated in

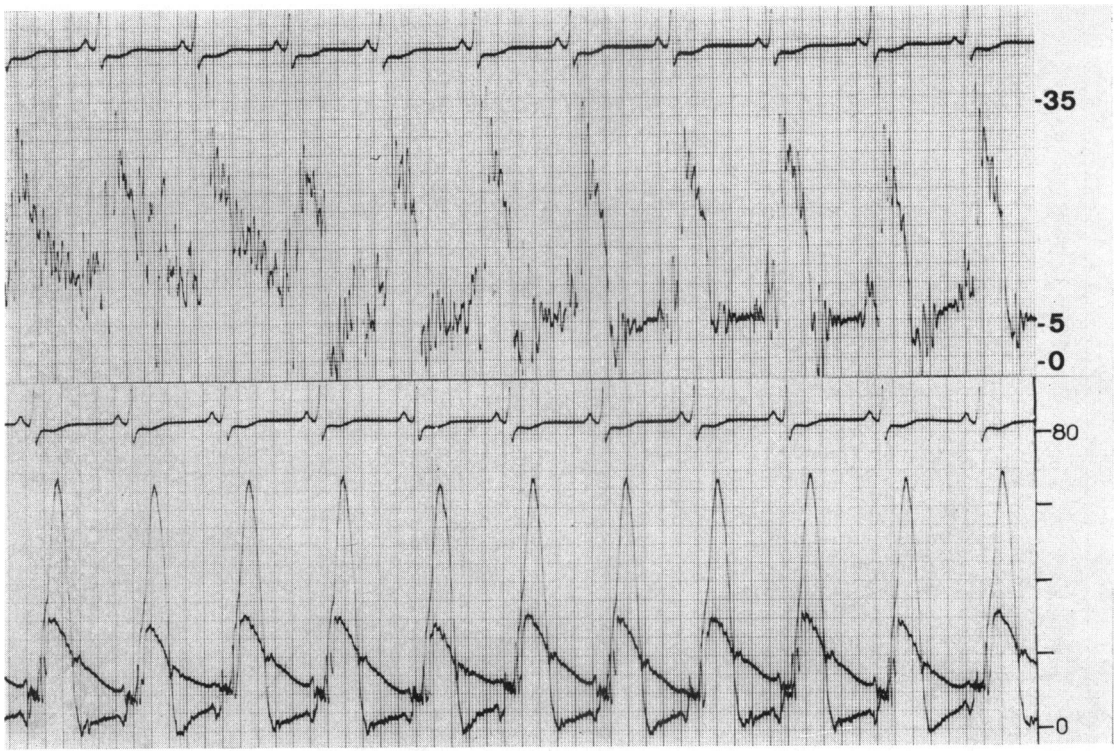

FIG. 2.—Case 11. Obstructive cardiomyopathy. *Upper half.* Record of pressures obtained during withdrawal of the catheter from the pulmonary artery to the right ventricular inflow tract. There is no evidence of infundibular stenosis. *Lower half.* Simultaneous records obtained from the pulmonary artery and the apex of the right ventricle. A systolic pressure difference is clearly demonstrated. Paper speed = 25 mm./sec. Pressures in mm. Hg.

three patients of this series (Table I, Cases 4, 8, 9): left ventricular involvement, in the absence of any significant pressure difference at rest inside the left ventricle, is demonstrated by the carotid pulse tracing, cine-angiocardiography, and isoprenaline infusion.

Factors Modifying Right Intraventricular Obstruction. Isoprenaline. The results of isoprenaline infusion are shown in Table IV. The systolic pressure in the pulmonary artery was unaffected. The systolic pressure in the apex of the right ventricle rose in all 4 patients. The peak systolic gradient was distinctly increased (Fig. 7). The variations in the pulmonary artery pressure did not appear to be linked with the effect of isoprenaline on the left heart since the pulmonary artery diastolic pressure (which is closely related to the "wedge"

TABLE IV

OBSTRUCTIVE CARDIOMYOPATHY: ISOPRENALINE STUDIES

Subject	Condition	Pulmonary artery		RV apex systolic (mm. Hg)	Systolic gradient (mm. Hg)	Cardiac index (l./min.m.^{2})	Stroke index (ml./beat m.^{2})
		Systolic	Diastolic				
6	Control	28	12	33	5	5.04	55
	Isoprenaline	40	11	62	22	7.04	54
7	Control	25	9	41	16		
	Isoprenaline	30	10	72	42		
8	Control	27	9	35	8		
	Isoprenaline	26	8	60	34		
9	Control	19	7	33	14	4.04	48
	Isoprenaline	12	4	40	28	7.84	68

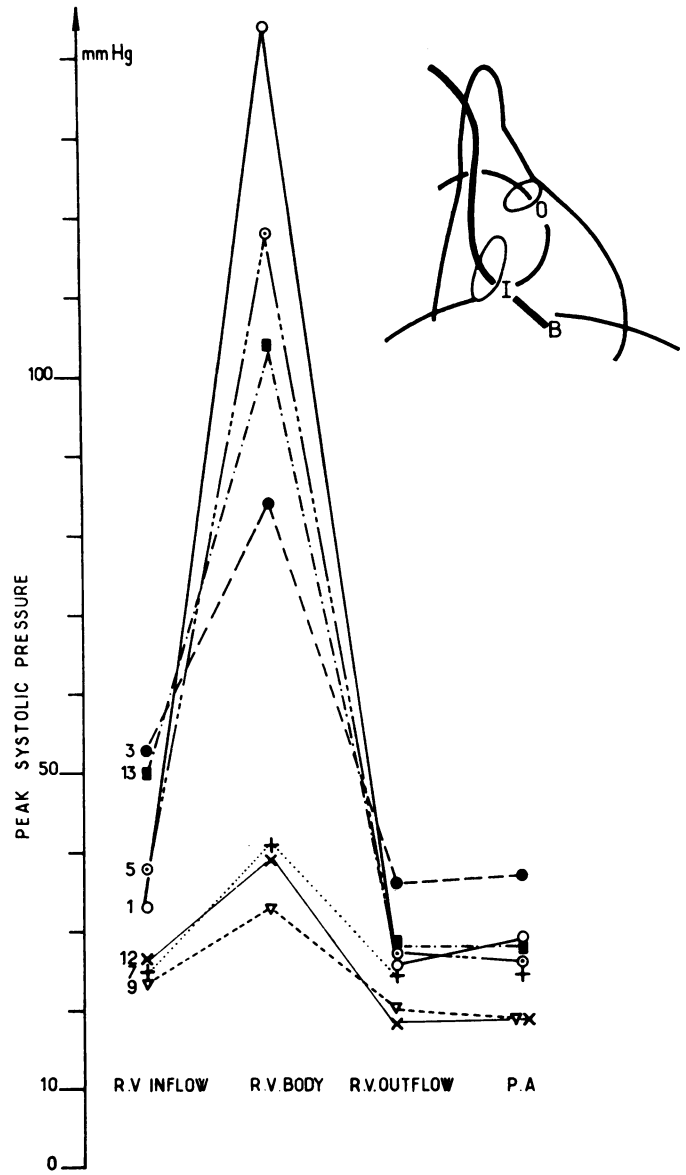

FIG. 3.—Plot of peak systolic pressures recorded, as shown in the right upper corner, from the pulmonary artery (PA), the apex (RV body (B)), the inflow tract (RV inflow (I)), and the outflow tract (RV outflow (O)) of the right ventricle. Each observation is represented by a different symbol and identified by the number on the left (Table III)

pressure) was unaffected. Likewise, the variations in pressure in the apex of the right ventricle appeared to be dissociated from those in the pulmonary artery and henceforth could not be considered as passive consequences of the latter. Increase in the left intraventricular gradient (Table I, Cases 7, 8, and 9) was simultaneous with that in the right intraventricular gradient and with the rise in cardiac output (Cases 6 and 9).

Propranolol (Inderal). Results of propranolol studies are listed in Table V. The peak systolic

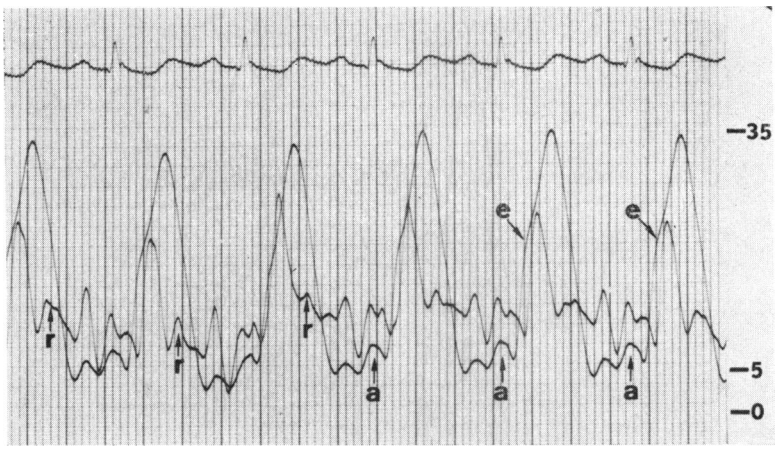

FIG. 4.—Simultaneous records obtained from the pulmonary artery and the apex of the right ventricle. Three distinct anomalies can be seen on the right ventricular pressure curve: (1) the notch on the ascending limb (e); (2) the tall "a" wave (a); (3) the raised end-diastolic pressure. The pulmonary artery pressure curve is abnormal: (1) The sharp mid-systolic descent is synchronous with the late systolic pressure rise in the right ventricle; (2) the late systolic peak (r). Paper speed = 50 mm. /sec. Pressure in mm. Hg.

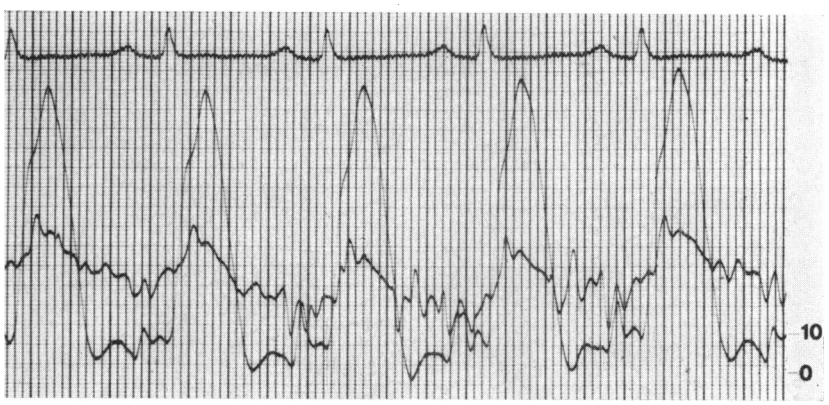

FIG. 5.—Simultaneous records obtained from the pulmonary artery and the apex of the right ventricle. Anomalies as in Fig. 4, except for the questionable mid-systolic trough on the pulmonary artery pressure curve. Pressures in mm. Hg.

gradient was lowered in one patient, and unaffected in the other (Fig. 7)*.

Post-ectopic Beats. The average pressure of several post-ectopic beats was compared to that of 10 normally conducted beats in Table V. The right systolic intraventricular gradient increased (Fig. 7). This was due to a pressure rise in the right ventricular apex, whereas the systolic pressure in the pulmonary artery (Cases 4 and 10) or the outflow tract (Case 12) was unaltered. Propranolol did not suppress this effect of post-extrasystolic potentiation (Fig. 8).

Intracavitary Asynchronous Pacing. Results of attempts to modify the intracavitary obstruction by asynchronous pacing are recorded in Table VI. The right intraventricular peak systolic pressure difference was reduced during stimulation applied to

* A third case has been studied during propranolol infusion after completion of the present study. The right ventricular systolic pressure was not altered (control: 33, propranolol: 32 mm. Hg); the pulmonary artery systolic pressure rose slightly (C: 17, I: 22 mm. Hg); the peak systolic gradient decreased (C: 16, I: 10 mm. Hg); the cardiac output fell from 2·56 to 2·15 l./min. m.^{2}.

TABLE V

OBSTRUCTIVE CARDIOMYOPATHY: PROPRANOLOL AND POST-ECTOPIC BEAT STUDIES

Subject	Condition	Pulmonary artery		RV apex systolic (mm. Hg)	Systolic gradient (mm. Hg)	Cardic index (l./min.m.²)	Stroke index (ml./beat m.²)
		Systolic	Diastolic				
				Propranolol			
11	Control	35	11	73	38	3·32	57
	Propranolol	32	10	43	11	2·94	53
12	Control	19	10	40	21	3·08	34
	Propranolol	22	10	42	20	2·36	30
				Post-ectopic			
4	Control	17	6	34	17		
	Post-ectopic	23	8	55	32		
10	Control	37	22	69	32		
	Post-ectopic	39	19	87	48		
12*	Control	18†		42	24		
	Post-ectopic	19†		85	66		

* Continuous infusion of propranolol throughout study.
† Right ventricular outflow tract pressures.

TABLE VI

OBSTRUCTIVE CARDIOMYOPATHY: ARTIFICIAL PACING STUDIES—CASE 10

Condition	Position of electrode catheter	Rate	Pressures (mm. Hg)*			Systolic gradient (mm. Hg)
			Pulmonary artery		Right ventricle apex	
			Systolic	Diastolic		
Sinus rhythm		72	25	7	99	74
Artificial pacing	Right ventricle apex	98	25	11	54	29
Sinus rhythm		69	25	12	97	72
Artificial pacing	Right ventricle apex	105	23	10	50	27
Artificial pacing	Left ventricle outflow	79	24	10	34	10
Fusion beats†	Left ventricle outflow	79	25	13	73	48

* Average values for 10 heart beats.
† Fusion beats resulting from interplay of sinus rhythm and artificial pacing.

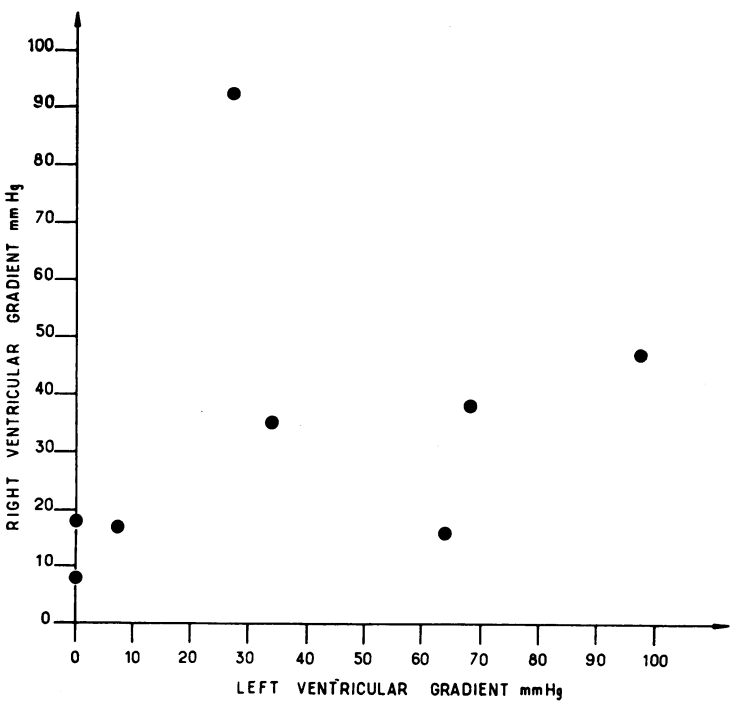

FIG. 6.—Obstructive cardiomyopathies studied in basal conditions. There is no correlation (r=0·09) between peak systolic pressure differences inside the left ventricle and inside the right ventricle.

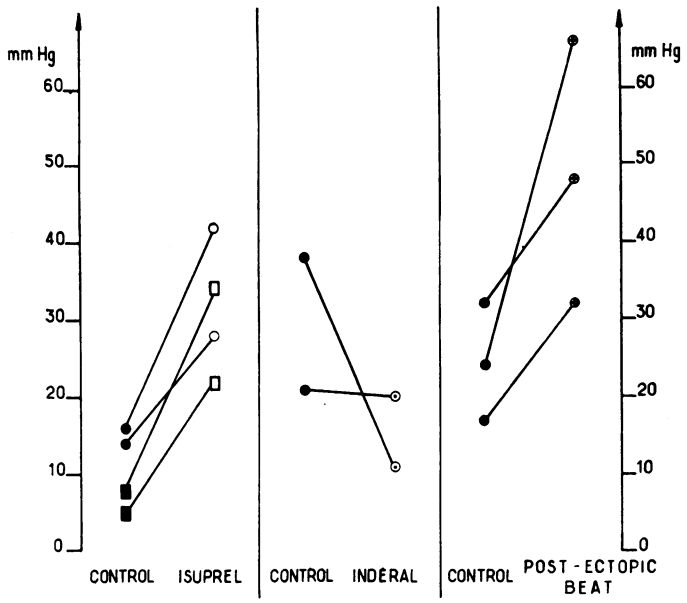

FIG. 7.—Alterations in right intraventricular obstruction under the influence of isoprenaline (*left*), propranolol (inderal) (*middle*), and post-extrasystolic potentiation (*right*). Each line is one observation and connects the values of the peak systolic pressure difference measured during the control period (*left*) and that observed during the given experiment (*right*)

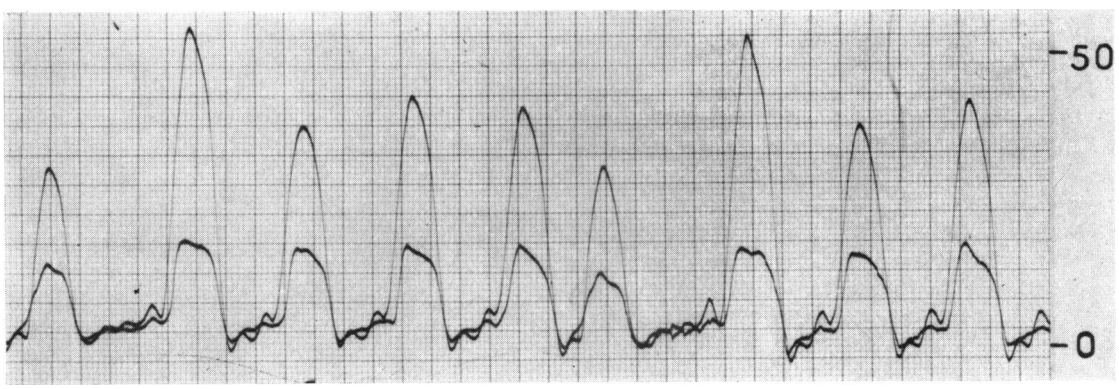

FIG. 8.—Simultaneous records of pressures obtained from the apex (*upper trace*) and outflow tract (*lower trace*) of the right ventricle during propranolol infusion. Starting from the left, beats 2 and 7 are post-ectopic beats. Pressures in mm. Hg.

TABLE VII
OBSTRUCTIVE CARDIOMYOPATHY: RIGHT VENTRICULAR GRADIENTS

Authors	Number of studies	Number of catheterizations of right ventricle apex	Number of gradients	Range of gradients (mm. Hg)
Soulié *et al.* (1962)	8	?	6	7–48
Wigle *et al.* (1962)	9	?	6	7–24
Bourdarias *et al.* (1964)	28	5	14	10–118
Braunwald *et al.* (1964)	59	?	10	10–61
Cohen *et al.* (1964)	24	?	13	10–66
Taylor *et al.* (1964)	4	2	3	27–78
Goodwin *et al.* (1964)	8	8	7	7–55

the right ventricular apex or to the subaortic chamber of the left ventricle. During left ventricular pacing alternate sequences of fusion beats and regular artificial pacing occur at identical rates. Both the right ventricular systolic pressure and the peak systolic pressure difference were close to control values during fusion beats whereas they fell during effective pacing.

Cine-angiocardiography of Right Ventricle. Opacification of the right ventricle has been achieved twice through selective injection of the dye, once through peripheral venous injection. The patient was rotated into the right anterior oblique position; the inflow and outflow tracts of the right ventricle had normal contours. However, some degree of obstruction was seen in Cases 5 and 12 at the junction of the inflow and outflow tracts during late systole. In the three cases the essential anomaly lies in the apical part of the ventricle. Diastolic filling was grossly impaired. During systole the apex disappeared almost completely. These findings suggested a prominent hypertrophy of the interventricular septum encroaching upon the lower part of the right ventricle.

Results of Control Studies. Results of the studies made in the control group are shown in Table II and Fig. 9. A significant pressure difference between the apex and the outflow tract of the right ventricle was found in two cases only. One was a case of patent ductus arteriosus in which this finding was not satisfactorily explained. The other was a case of bilocular right ventricle associated with pulmonary valvular stenosis.

Discussion

The incidence and degree of the right intraventricular systolic pressure difference in a number of obstructive cardiomyopathies collected from published reports are shown in Table VII. Both are closely related to the care taken to explore not only the inflow and outflow tracts but also the apex of the right ventricle. The incidence of right ventricular obstruction rises from 13 of 24 (Cohen *et al.*, 1964) to 7 of 8 (Goodwin *et al.*, 1964), or from 14 of 28 (Bourdarias *et al.*, 1964) to 11 of 12 in the present study, when pressure recording from the apex of the right ventricle has been systematic. This fact has been emphasized lately by Taylor *et al.* (1964). The right intraventricular obstruction cannot be considered as a low infundibular stenosis since a gradient between the inflow and outflow chambers of the right ventricle is infrequent, as shown in the present study, or usually moderate (Braunwald *et al.*, 1964; Bourdarias *et al.*, 1964).

The high pressures recorded from the apex of the right ventricle are not due to an artefact, and our data show that a significant pressure difference between the apex and the remainder of the right ventricle is quite uncommon in other conditions involving the right ventricle.

Cine-angiocardiography of the right ventricle confirms the exclusion of the apex suggested by the pressure tracings. In three cases of this study and in three other cases previously studied in the same laboratory (Ferrané *et al.*, 1964), the apex of the right ventricle is strikingly reduced, almost blotted out by the hypertrophied myocardium during systole and, to a lesser extent, during diastole. The contrast medium can be seen to flow freely from the right atrium to the pulmonary artery through the upper part of the right ventricle. Similar findings have been made during angiocardiography (Cohen *et al.*, 1964; Steiner, 1964; Oakley, 1964).

The nature of the right ventricular obstruction deserves discussion. Evidence in favour of a functional dynamic stenosis is controversial. The pharmacodynamic studies mentioned above suggest that it is present. First, isoprenaline brings about a rise in the intraventricular systolic pressure difference in the absence of changes in pulmonary artery systolic and diastolic pressures. Secondly, post-extrasystolic potentiation is accompanied by a rise in pressure in the apex of the right ventricle, whereas the pressure is unaltered in the pulmonary artery or outflow tract. These findings are in agreement with observations made by Wigle (1964b) and Braunwald (1964), but are at variance with Goodwin *et al.*'s (1964) experiments with isoprenaline, amyl nitrite, and phenylephrine and with Oakley's (1964) measurements during post-ectopic beats. These authors state that "the lack of any significant change in gradient on the right side of the heart, the parallel increase in right ventricular and pulmonary artery pressures . . . all suggest that the right heart is not primarily involved in the disorder of contraction but is affected passively by the gross generalized hypertrophy especially of the septum, which is such a constant feature" (Goodwin *et al.*, 1964). However, the lack of correlation between the degree of the left and right intraventricular gradients (Fig. 6) does not support the conclusion that the right-sided obstruction can be merely explained on a mechanical basis.

The dynamic disorder of contraction is superimposed on the right ventricle anatomical anomalies. This is clearly shown by angio- or cine-angiocardiographic studies (Cohen *et al.*, 1964; Steiner, 1964; Oakley, 1964), by necropsy specimens, and by surgical findings (Taylor *et al.*, 1964).

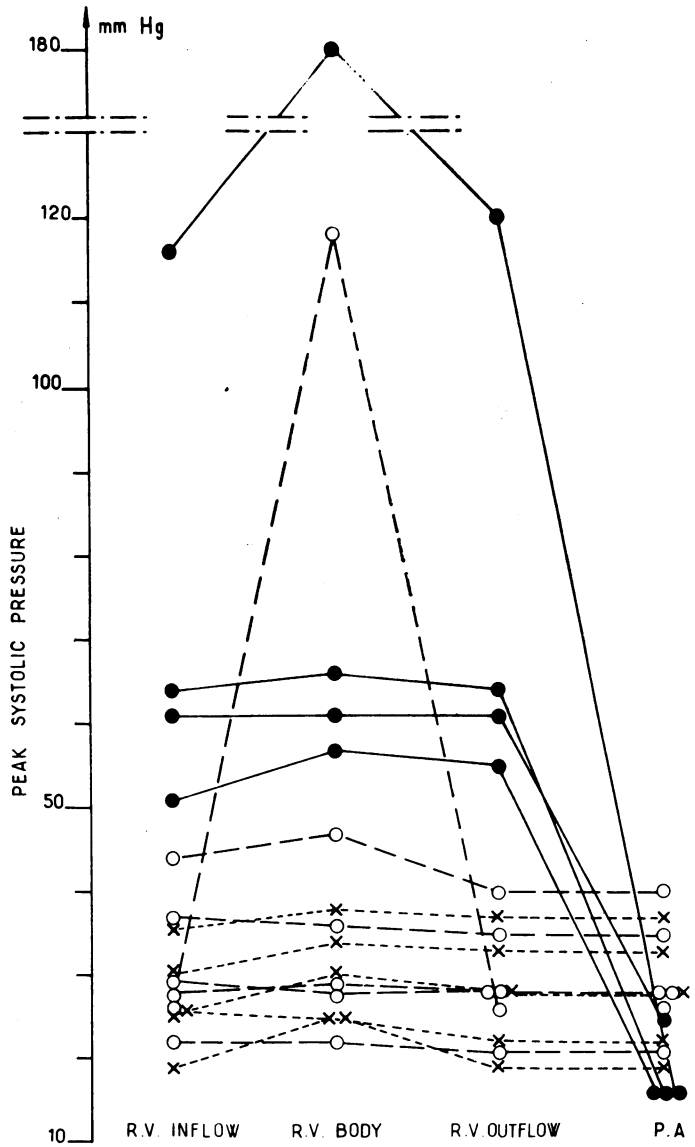

FIG. 9.—Plot of peak systolic pressures in the series of 15 control patients. Abbreviations as for Fig. 3. Pulmonary valvular stenosis is indicated by full circles and heavy lines; left-to-right intracardiac [shunts, by open circles and broken lines; and miscellaneous, by crosses and dotted lines. A significant pressure difference between the apex and remainder of the right ventricle is found in two cases only, one with a bilocular right ventricle.

The mechanism of the dynamic process involved in the intraventricular obstruction is poorly understood. Beta-adrenergic inhibitors such as pronethalol (Goodwin *et al.*, 1964) and propranolol in the present study do not reduce significantly the right intraventricular obstruction. This suggests that the latter is not related to an enhanced activity of the beta-adrenergic receptors. The artificial pacing study conducted in one patient in the present series suggests that the sequence of activation of the right ventricle plays a part in the obstruction. At an identical heart rate the obstruction was more severe during fusion beats occurring during the left ventricle pacing than it was during completely

driven beats. More experiments are necessary before any further conclusion can be drawn on this point.

Summary

Pressure gradients in the right ventricle have been studied in obstructive cardiomyopathy and in another group of patients. In the former, studies were made at rest, during infusion of isoprenaline and propranolol, and after asynchronous pacing. In obstructive cardiomyopathies, the results suggest that right ventricular obstruction is constant and is due to both muscular hypertrophy and a dynamic disorder of the contraction.

A severe degree of obstruction is seldom found between the inflow and outflow tracts of the right ventricle, whereas there is considerable evidence that the obstruction predominates in the apex of the right ventricle. Diastolic filling of this portion of the ventricle is grossly restricted. Pressure records and cine-angiocardiography demonstrate that the apex is "cut-off" from the upper part of the ventricle during systole.

Further studies are necessary for a proper evaluation of the involvement of the right ventricle in determining the signs and symptoms of obstructive cardiomyopathy and the therapeutic approach to the disease.

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